

## COVID-19 coagulopathy vs disseminated intravascular coagulation

Marcel Levi

Department of Medicine, University College London Hospitals (UCLH) NHS Foundation Trust, London, United Kingdom; and Cardiometabolic Programme, Biomedical Research Centre, National Institute for Health Research, UCLH/University College London, London, United Kingdom

The audio version of this Blood Advances Talk is available on the full-text article page.

## **Abstract**

Patients with severe COVID-19 infections frequently manifest coagulation abnormalities that are associated with respiratory deterioration and death. In addition, many patients with severe COVID-19 infections develop thromboembolic complications, which seem to be related to the coagulopathy. It has been suggested that undiagnosed pulmonary embolism contributes to a sudden deterioration of pulmonary oxygen exchange that is sometimes seen in patients with COVID-19 infections. The coagulation changes associated with COVID-19 mimic other systemic coagulopathies that are regularly seen during severe infections, such as disseminated intravascular coagulation or thrombotic microangiopathy. However, at the same time, the clinical and laboratory characteristics of the coagulation changes in COVID are distinctly different from those in the common presentation of these conditions. Severe COVID-19 infections seem to cause a profound coagulation abnormality caused by inflammation-induced changes in coagulation in combination with severe endothelial cell injury, with consequent massive release of von Willebrand factor and plasminogen activators. This coagulopathy likely contributes to pulmonary microvascular thrombosis, bronchoalveolar fibrin deposition (which is a hallmark of adult respiratory distress syndrome), and thromboembolic complications.

Download or subscribe to the *Blood Advances* Talks podcast at https://soundcloud.com/blood-advances.

The complete text of this *Blood Advances* Talk is available as a data supplement. © 2020 by The American Society of Hematology

Contribution: M.L. performed the literature review and wrote the paper.

Conflict-of-interest disclosure: The author declares no competing financial interests. ORCID profile: M.L., 0000-0002-2212-5299.

Correspondence: Marcel Levi, University College London Hospitals, 250 Euston Rd, London NW1 2PG, United Kingdom; e-mail: marcel.levi@nhs.net.